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WHAT IS THE OUTLOOK FOR PATIENTS WITH VENOUS LEG ULCERS?

Microbiology

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ABSTRACT

Venous ulcer as an open skin lesion of the leg or foot that occurs in an area affected by venous hypertension. Venous ulcers are wounds on the lower legs brought about by chronic venous insufficiency. Due to blood amalgamate inside the vein, pressure assembly bursts open fluid to ooze out into neighborhood tissues .The cause of venous ulcers is high pressure in the veins of the lower leg. The excess fluid that annoys the tissue leads to inflammation and, inturn, tissue conks out. The resulting wound is slow-healing and will often recur. Venous ulcers can occur when the veins in patient legs do not push blood back up to the heart. Blood backs up in the veins, building up pressure. If not treated, increased pressure and excess fluid in the affected area can cause an open sore to form. These wounds can last and occur again for years, significantly impacting quality of life. Venous leg ulcers are late manifestations of chronic venous insufficiency that can cause impairment and serious complications. Venous leg ulcers (VLUs) often affects patient care, healing time, and quality of life. **Aim:** Venous insufficiency (also called chronic venous insufficiency or chronic venous disease) is a condition in which the leg veins are damaged, preventing normal blood flow to the heart. In normal veins a series of specialized, one-way valves work together, opening to allow blood to flow upward and then closing to keep the blood from flowing back toward the feet. Venous insufficiency occurs when valves are damaged or not functioning properly. As the valves deteriorate, blood leaks or flows backward and pressure in the vein increases, stretching and dilating the vessel. Blood pools in the veins of the lower legs, increasing the blood pressure in the legs and causing chronic inflammation in the veins.

KEYWORDS

Venous leg ulcers (VLUs), venous insufficiency (CVI) , venous hypertension

INTRODUCTION

Venous leg ulcers are defined as open lesions between the knee and ankle joint that occur in the presence of venous disease resulting in reduced mobility, poor quality of life, and financial implications. (1)

Skin substitutes and skin grafts can be used as a primary modality for large ulcers more than 25cm (2).Lipodermatosclerosis is a type of panniculitis characterized by pronounced skin induration and inflammation (3).

Infiltration of the primary inflammatory cells such as macrophages, lymphocytes, and plasma cells in the tissue site, producing inflammatory cytokines, growth factors, enzymes (4,5)

Lymphocytes including T-lymphocytes and B-lymphocytes are the next line of defense, and they play a crucial role in mediating inflammation by several complex mechanisms including secreting of cytokines, co-stimulation of lymphocytes, and production of antibodies and immune complexes.(6,7)

This is defined as an abnormally functioning venous system caused by venous valvular incompetence. Venous outflow may or may not be obstructed, and the abnormal function may affect the superficial venous system, the deep venous system, or both (8).

The development of this condition is influenced by multiple factors, including genetics, female sex, pregnancies, age, prolonged standing, trauma, and obesity. Some of these factors can be mitigated through lifestyle (increasing exercise, controlling body weight, and avoiding smoking), but others are not modifiable and many individuals will inevitably develop CVD over time. (9)

This condition is diagnosed based on history, clinical presentation, and diagnostic tests, with duplex ultrasound being the gold standard (10). Clinical assessment should describe the ulcer area, depth, edges, wound base, signs of infection, and peripheral skin changes (11). The proinflammatory microenvironment is maintained by M1 macrophages, mainly through the release of IL-1α, IFN-γ, and TGF-β1.(12) The majority of risk factors for the development of VLUs are non-modifiable, and patients often present more than one. These involve a family history of CVI, advanced age, female sex, previous thrombosis or pulmonary embolism, multiparity, lipodermatosclerosis, musculoskeletal and joint disease (13)

Modifiable risk factors are obesity and sedentarism are also associated with venous disease (14)

This venous hypertension in the larger veins is transmitted to the skin's microcirculation, contributing to chronic changes in the soft tissue and skin. (15)

Treatment guidelines support compression therapy as the primary therapeutic modality for decreasing reflux and healing VLUs.(16,17) However, many wounds remain unhealed with high recurrence rates. Recently, the Early Venous Reflux Ablation (EVRA) trial demonstrated that early endovenous ablation of superficial venous reflux reduces the time to VLU healing, increases ulcer-free time, and is likely to be cost effective.(18)

Sclerotherapy may target the terminal reflux or eliminate venous hypertension of reservoir vessels in the small venules at or near the ulcer bed. Small studies have also shown foam sclerotherapy to improve ulcer healing rates and to decrease recurrence rates over compression therapy alone (19)

Venous ulcers are wounds that are thought to occur due to improper functioning of venous valves, usually of the legs (20)

Other names to Venous insufficiency ulceration are stasis ulcer,/ stasis dermatitis/, varicose ulcer/, ulcus cruris/, and crural ulceration

"Rising levels of obesity mean that the number of people who suffer from leg ulcers is likely to grow."

Smoking, Obesity, Older age, Paralysis, Sedentary lifestyle with limited physical activity, Deep vein thrombosis. Family history of venous disease, previous injury. Surgery, such as a knee replacement and varicose and spider veins.

Risk Factors

Risk factors for venous ulceration were age, male sex, personal history of superficial and deep venous thrombosis, diabetes, high blood pressure, skeletal or joint disease in the legs and emphysema or chronic obstructive pulmonary disease, higher body mass index and physical inactivity, parental history of ankle ulcer as well as reflux in deep and

perforator veins, deep obstruction, and combination of reflux and obstruction.

Factors Affecting The Size Of Venous Ulcers

Superficial venous insufficiency is defined as a prolonged duration of retrograde flow on ultrasound leading to Venous hypertension .Venous hypertension can manifest in a wide range of clinical spectrum which includes dilated veins , reticular veins , telangiectasias, varicose veins, to Stasis eczemas finally resulting in venous leg ulceration.

Venous leg ulcers are defined as open lesions between the knee and ankle joint that occur in the presence of venous disease[1] resulting in reduced mobility, poor quality of life, and financial implications.

PATHOPHYSIOLOGY

Venous leg ulcers are common in people with varicose veins or mobility problems whose "muscle pumps" in the feet and calves struggle to drive blood up to the heart. These ulcers can be painful and unsightly, having a significant negative impact on health and quality of life. Chronic venous insufficiency is the result of a complex interplay between genetic and environmental factors, causing abnormalities in the venous macrovasculature, microvasculature, skin, and soft tissue, often complicated by chronic infection and inflammation.

Factors associated with venous incompetence are age, sex, family history of varicose veins, obesity, phlebitis, previous leg injury, and prolonged standing or sitting posture.

Venous hypertension, the major contributory factor, results from, superficial and deep vein insufficiencies, Deep vein thrombosis, and insufficiencies of calf muscle pumps and perforators. The concomitant increase in venous hypertension can lead to the anatomic, physiologic, and histologic changes associated with chronic venous disease.

Elevated intravascular pressure leads to formation of The "fibrin cuff" around the dermal capillaries. This decreases oxygen permeability 20-fold. Leading to tissue hypoxia causing impaired wound healing. This fibrin cuff forms a niche for uncontrolled influx of inflammatory cells which leads to dysregulation of various pro-inflammatory cytokines and growth factors like tumor necrosis factor-α (TNF-α), TGF-β, and matrix metalloproteinase.[21]

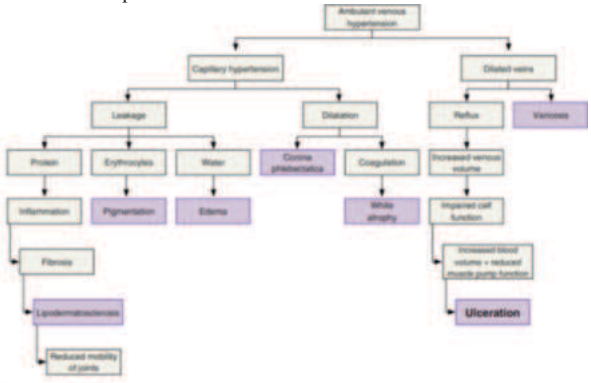


Figure 2 The Rotterdam model explains the pathways from venous hypertension to venous leg ulcer (clinical symptoms are in purple).

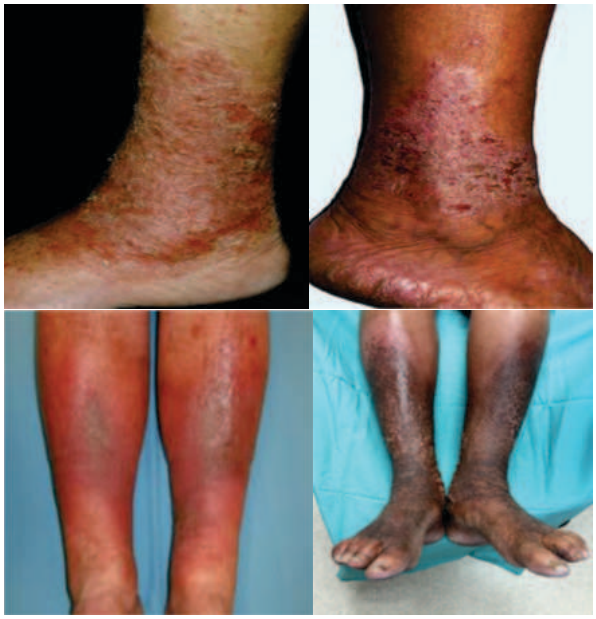
CLASSIFICATION

Robles-Tenorio A, Lev-Tov H, Ocampo-Candiani J. Venous Leg Ulcer et al

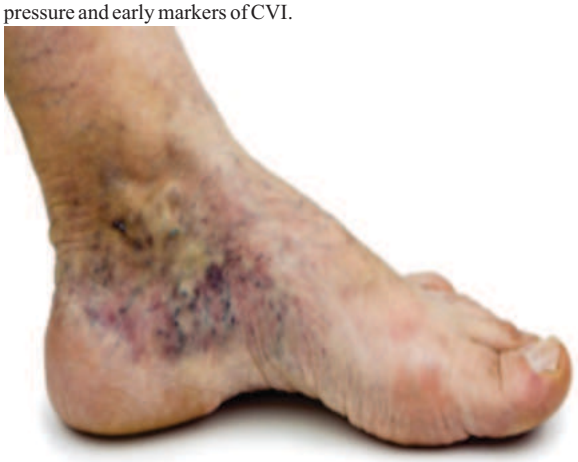
Clinical	
C0	No visible or palpable signs of venous disease
C1	Telangiectases or reticular veins
C2	Varicose veins
C3	Edema
C4a	Pigmentation or eczema
C4b	Lipodermatosclerosis or atrophie blanche
C5	Healed venous ulcer
C6	Active venous ulcer
Etiological	
Ec	Congenital
Ep	Primary
Es	Secondary (post-thrombotic)
En	No venous cause is identified.
Anatomical	
As	Superficial veins
Ap	Perforator veins
Ad	Deep veins
An	No venous location was identified.
Pathophysiological	
Pr	Reflux
Po	Obstruction
Pr, o	Reflux and obstruction
Pn	No venous pathophysiology identifiable

Skin Changes In Venous Insufficiencies

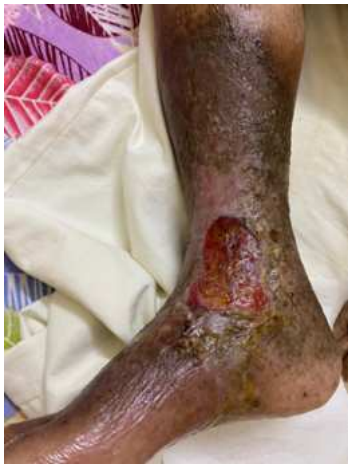
Skin can play a major contribution to early diagnosis before chronic venous diseases progress further. The most common skin finding after varicose veins is stasis dermatitis. It was observed in 32.7% of the patients as one of the early findings of chronic venous insufficiency (CVI). It occurs as a result of increased venous pressure in the lower extremities characterized by erythema, eczematous patches, and plaques with unclear borders in the lower extremities and around the medial malleolus.



Blue telangiectasias and intradermal venules on the medial side of the ankle and foot are considered to be a chronic sign of high distal venous pressure and early markers of CVI.

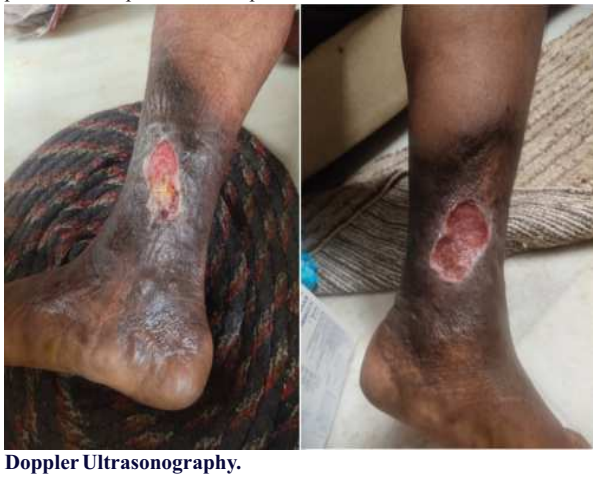


CVI causes varying degrees of skin pigmentation on the leg. Lipodermatosclerosis[3] is a type of panniculitis characterized by pronounced skin induration and inflammation. The skin hardens and thickens and may also be accompanied by edema, pigmentation, blanch atrophy, and ulcers. The leg looks like an inverted bottle. Atrophie Blanche is plaques that exhibit porcelain white, star-shaped atrophic scars exhibiting telangiectasia and hyperpigmentation in the periphery of white atrophy. Early referral by the Dermatologists to the surgeons can aid in early detection and management.



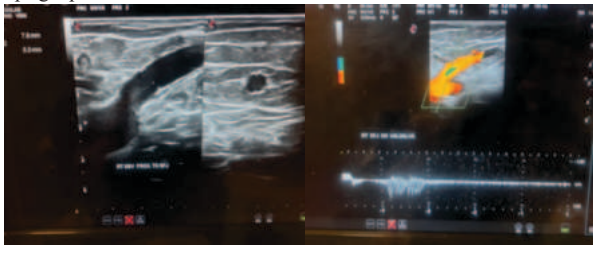
EVALUATION

1. Clinical inspection includes evaluating the area of ulcer, its depth, edges, wound base, signs of infection, and peripheral skin changes.
2. Palpation of distal pulses and ankle-brachial pressure index (ABPI) is of paramount importance. ABPI > 1.3 indicates significant vascular calcification needing urgent referral to a vascular specialist
3. Color-flow duplex ultrasound is a highly informative diagnostic tool particularly useful for superficial vein assessment which can identify thrombi presence and valve incompetence
4. Computed tomography and magnetic resonance imaging can be used to diagnose abnormal deeper vessels
5. Photoplethysmography is a noninvasive test that measures venous refill time to determine the efficiency of the calf muscle pump and the presence of any abnormal venous reflux. Patients who have poor emptying of the veins and abnormally rapid refilling (<25 s) can have problems in superficial or deep veins.



Doppler Ultrasonography.

Leg ulcers caused by venous diseases are effectively assessed by Doppler ultrasonography. The examination provides clear anatomical and physiological information for the diagnosis, treatment planning, and real-time guiding during the surgical treatment. Diagnostic Doppler ultrasonography assesses deep, superficial, and perforator veins, starting from patency assessment by direct visualization and simply compression test. The internal flow can be assessed by pulse wave analysis, which is used for rule out downstream flow obstruction and valvular incompetence. The venous valve function of deep, superficial, and perforator systems can be evaluated by measuring the time of the retrograde flow after flow augmentation performing in the upright position.



MANAGEMENT

Principles Of Management

1. To reduce edema
2. To counteract venous hypertension
3. To increase venous blood return
4. To include venous interventions to treat the underlying venous pathology
5. Enhance wound /ulcer care
6. To reduce ulcer recurrence.

CONSERVATIVE MANAGEMENT

1. Compression therapy

First line treatment for patients with chronic venous insufficiency and varicose ulcers. It decreases venous hypertension, which enhances wound healing and decreases recurrence.

Multicomponent systems show better results than single-component systems, and elastic therapies are preferable over nonelastic treatment The most standard form of compression therapy are Compression stockings or compression hosiery with full foot, knee high, 30 to 40 mmHg being the most commonly prescribed ones.

Used one hour per day, at least six days per week to reduce recurrence.

Leg elevation and supervised exercise programs to decrease venous pressure and limb oedema.

No role of diuretics

2. Dressings

Dressings are recommended to cover ulcers and promote moist wound healing. Dressings should be selected based on wound location, size, depth, moisture balance, presence of infection, allergies, comfort, odor management, ease and frequency of dressing changes, cost, and availabilityref (22)

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3.

a) Systemic therapies

• Anti-inflammatory and antithrombotic agents :

Many anti-inflammatory drugs like steroids, aspirin, ifetroban, and others have failed to show benefit in ulcer healing compared with placebo.

• Pentoxifylline –used as an adjunct to compression

• Antimicrobial therapy:

When infection is suspected clinically, a tissue specimen should be sent for gram stain, culture, and sensitivity as a guide to select appropriate antimicrobial therapy. Systemic antibiotics are indicated for patients with clinical evidence of infection and with >100,000 colonizing forming units (CFU)/g in the wound bed. Routine use of antibiotics is not recommended

b) Topical Agents:

Topical agents are used selectively to avoid contact sensitization and Allergic contact dermatitis. Hydrogel with or without silver, collagenase, honey, povidone-iodine (Betadine), peroxide-based preparations can be tried. Silver sulfadiazine must be used with caution as it can cause contact sensitization

II. DEBRIDEMENT

Debridement involves the removal of necrotic tissue to enhance wound healing. Debridement may be sharp, enzymatic, mechanical, larval, or autolytic out of which Sharp and Enzymatic debridement using collagenase have shown effective results

III. SURGICAL MANAGEMENT

a) Endo venous ablation: Endo venous ablation, ligation, subfascial

endoscopic perforator surgery, and sclerotherapy are different modalities showing promising results in early stages in conjunction with compression therapy

b) Skin substitutes and skin grafts: Skin substitutes and skin grafts can be used as a primary modality for large ulcers more than 25cm and play a secondary role for ulcers that have failed to heal after maximizing medical care after a period of four to six weeks. Split skin thickness grafts can be tried after the Wounds are appropriately debrided with bacterial overgrowth under control. Importance to the management of peri ulcer area is also to be given.

POOR PROGNOSTIC SIGNS

1. Advanced Age
2. Elevated Body Mass Index
3. Ulcer Size of 10 Cm Or More
4. Ulcer Duration Longer Than Three Months
5. Presence of Lower Limb Arterial Disease

PREVENTION OF ULCER RECURRENCE

1. Continued use of graduated compression hosiery
2. Leg elevation and exercise to improve calf muscle pump function
3. Repeat venous duplex ultrasound to identify any segments with incompetence

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